THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI
2010

A Dissertation on

A STUDY OF CEREBROSPINAL FLUID DYNAMICS IN COMMUNICATING HYDROCEPHALUS

Submitted in partial fulfillment of
the requirements of

M.Ch., Branch II, (Neurosurgery - 5 years course)
Examinations August 2010

INSTITUTE OF NEUROLOGY
MADRAS MEDICAL COLLEGE
CHENNAI – 600 003

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF CEREBROSPINAL FLUID DYNAMICS IN COMMUNICATING HYDROCEPHALUS"** submitted by **Dr. Vidhya Narasimhan** appearing for **M.Ch.** Degree examination in **August 2010** is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I, Dr. Vidhya Narasimhan solemnly declare that this dissertation **"A STUDY OF CEREBRO SPINAL FLUID DYNAMICS IN COMMUNICATING HYDROCEPHALUS"** was prepared by me at the Institute of Neurology, Madras Medical College and Government General Hospital, Chennai under the guidance and supervision of Professor of Neurosurgery, Institute of Neurology, Madras Medical College and Government General Hospital, Chennai between 2006 and 2010.

This dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the University requirements for the award of degree of M.Ch. Neurosurgery.

Place: Chennai

Date: **(Vidhya Narasimhan)**

ACKNOWLEDGEMENT

I owe my thanks to the Dean, Madras Medical College and Government General Hospital, Chennai, for permitting me to use the clinical material in the study.

I am greatly thankful to the Head of the Department and The Professor of Neurosurgery Prof. V.SUNDAR for allowing me to conduct this study .He was a constant source of encouragement throughout the study and ensured successful completion of this study.

I sincerely thank Prof. V.G.RAMESH, Professor of Neurosurgery for his expert guidance and valuable advice throughout the study.

I immensely thank Professors of Neurosurgery, Prof. K.DEIVEEGAN, Prof. R.ARUNKUMAR, Prof. K.MAHESHWAR, Prof. C.SEKAR , Prof. J.V.MAHENDRAN for their continuous support and encouragement to do the study.

I thank all the assistant professors, my fellow post graduates and the staff of the Institute of Neurology for their help in the study.

I thank the Statistician Miss. JAYANTHI for helping me to complete the study.

Last but not the least I would like to thank all the patients who participated in the study, whose full co operation has made the study possible.

CONTENTS

INTRODUCTION

Disorders of cerebrospinal fluid circulation often pose a challenge in diagnosis and management decisions. In patients with communicating hydrocephalus like normal pressure hydrocephalus, post meningitic or post traumatic hydrocephalus, selection of patients who are likely to improve with cerebrospinal fluid shunting procedures is still more a challenge. In communicating hydrocephalus there is defective absorption of cerebrospinal fluid. Hence studies of dynamics of cerebrospinal fluid circulation, like measurement of opening pressure, pressure volume index and outflow resistance would be of help in establishing the diagnosis, assessing the prognosis and predicting the outcome after management.

There are various methods of cerebrospinal fluid dynamics studies. In this study we have used improvised bolus lumbar injection method using bed side saline manometer, devised at our institute, 'The Madras Institute of Neurology (MIN) method'. It is a modification of Marmarou's bolus lumbar injection method by replacing the pressure transducers by simple saline manometer made with easily available bed side materials.

AIM OF THE STUDY

- To measure the opening pressure (P_o), pressure volume index (PVI), and cerebrospinal fluid outflow resistance (R_{out}), in patients with communicating hydrocephalus by bolus lumbar injection method.

- To evaluate the diagnostic and prognostic value of cerebrospinal fluid opening pressure and outflow resistance measurement in communicating hydrocephalus

- To formulate the criteria for selection of cases for shunting in communicating hydrocephalus

REVIEW OF LITERATURE

The review of literature is done under the following headings:

I. Cerebrospinal fluid (CSF) physiology

II. Various methods of studying cerebrospinal fluid dynamics

III. CSF dynamics in abnormal states and review of various studies cited in literature

I. Cerebrospinal Fluid Physiology

Cerebrospinal fluid dynamics includes all factors concerning its formation, circulation and absorption and the factors determining the CSF flow rate, direction and pressure.

Cerebrospinal Fluid Formation

The main production site for cerebrospinal fluid is the choroid plexus in the lateral, third and fourth ventricles by ultra filtration of plasma across the endothelial capillary wall, active transport of sodium and bicarbonate by the choroidal epithelium

The small extra choroidal part of cerebrospinal fluid production begins as interstitial fluid seeping away from brain either transependymal to the ventricles or transpial to the intracranial and intraspinal

subarachnoid spaces. The cerebrospinal fluid formation rate (Fr) is 0.35ml/min, 20ml/hour, total of 500ml/day. The total cerebrospinal fluid volume is 100-150 ml out of which the ventricular system contains 15-25 ml normally. A low cerebral perfusion pressure or long standing raised intra cranial pressure leads to decreased cerebrospinal fluid formation rate.

Cerebrospinal Fluid circulation

Cerebrospinal fluid leaves the ventricular system by paired foramen of lushka and midline foramen of magendie and circulates either down into the spinal subarachnoid space or passes the cisterns, - cistern magna, pontine, interpeduncular, ambient, suprasellar cisterns and makes up over the brain in the sulci to the arachnoid villi. This is called cerebrospinal fluid bulk flow.

In the arachnoid villi there is a layer of avascular arachnoid cells in the subarachnoid side, completely invested by an intact non fenestrated layer of endothelial cells continuous with the endothelial lining of superior sagital sinus. The villous cells have giant vacuoles and cerebrospinal fluid flows by transcellular transportation maintained by the pressure gradient between the subarachnoid space and the venous space of sagital sinus. The main driving forces for maintaining the flow of the

cerebrospinal fluid are arterial systolic pressure pulsation of the brain and probably the pressure gradient created by continuous CSF secretion.

Cerebrospinal Fluid absorption

The principle sites of cerebrospinal fluid absorption is through the villous valves penetrating through the venous sinusess by either valvular or vacoular mechanism. At normal steady state of intracranial pressure (ICP), CSF is transported across the endothelium by micropinocytic vesicles and inter-endothelial clefts, whereas at sustained raised ICP, an increased flow of CSF occurs by open transcellular channels.

To some extent absorption also occurs at

- Villi along the spinal roots,
- Along the brain capillaries of the subarachnoid space into the brain,
- Through the ependyma,
- Through the choroid plexus,
- Along the olfactory nerves into the nasal mucosa, then into the lymph system,
- Directly into the extracellular dura.

.

The pressure in cerebrospinal fluid space is higher than in sagital sinus which is higher than in torcular area and higher than the pressure in jugular vein.

Intracranial pressure (ICP) is related to dural sinus pressure (Pss) by the formula

$$Pss = 0.36ICP + 36\ mmH_2O$$

Regulation of CSF formation, absorption and ICP

The balance between CSF formation and absorption in holding the ICP constant is given by the parson equation-

$$ICP = F_r R_{out} + P_{ss}$$

$$= E_r R_{out} + P_{ss}$$

(E_r – CSF elimination rate)

The relation between formation rate and ICP is

$ICP = 3.0 + 0.3R_{out}$ as normal F_r is 0.3 ml/min

Monro-Kellie doctrine

The sum of the intracranial volumes, - cerebrospinal fluid, cerebral blood volume, interstitial fluid, neurons and glia – is constant.

An increase in cerebrospinal fluid (hydrocephalus) presupposes a reverse volume change in one of the other intracranial volumes. Distension of the spinal meninges, compression of venous vascular

structures, and venting of cerebrospinal fluid constitute the three major mechanisms which serve to protect the brain from elevations of ICP.

Regulation of cerebrospinal fluid volume by means of outflow resistance factors especially the venous part is probably the major mechanism for protection of the brain against the lethal increases of ICP.

Human beings have the highest overall rate of formation, the greatest efflux capacity, the lowest outflow resistance. Cerebrospinal fluid outflow can sustain an efflux rate of at least 2ml/min.

II. Measurement of resistance to cerebrospinal fluid outflow

The resistance to cerebrospinal fluid outflow can be measured by infusion or perfusion techniques, bolus injection methods and isotope dilution methods.

These methods monitor ICP during infusion of synthetic cerebrospinal fluid or ringer lactate. Three different techniques have been described.

- Constant pressure servo controlled infusion method
- Constant infusion method
- Constant infusion and constant pressure method

The basic principle underlying all the methods is infusing artificial cerebrospinal fluid intrathecally either at a constant rate or a constant pressure and plotting the flow in ml/min against the ICP levels. The slope

of the regression curve is the conductance to CSF flow (C_{out}), and the reciprocal value is the resistance to outflow (R_{out})

$$R_{out} = 1/C_{out}$$

The calculation of R_{out} implies a constant rate of CSF production and constant CSF and cerebral blood volumes, irrespective of the increase in ICP during the study.

All methods have the possibility of CSF leakage, which can result in low R_{out} values. The interpretation of pressure increase during the constant rate of lumbar infusion is difficult and unreliable. The closed system of some of the infusion methods makes spontaneous ICP fluctuations unavoidable.

Constant pressure servo controlled method

By this method R_{out}, P_{ss} and Pressure Volume relationship can be calculated but not $F_{r.}$ The interventional character and practical difficulties in constant intrathecal infusion has limited its use.

Constant infusion method (Katzman test)

ICP is monitored during constant infusion and from the pressure increase, the R_{out} can be calculated. It can be done in two methods:

Plain infusion test – it is easy and not very time consuming. Criteria for normal and abnormal curves are given, but the interpretation of the plateauing of the induced pressure rises, is difficult

Computerised infusion test – by computerised analysis of the ICP signal a more exact filtration of normal fluctuation is possible. The analogue pressure signal from the pressure amplifier is processed in a computer. From the non- linear regression curve of ICP during infusion static measurement of R_{out}, Fr, and Pressure Volume Index (PVI) are calculated. This method is faster than the other two infusion methods.

Constant infusion and constant pressure (Lumboventricular perfusion) method

It is a practical not too time consuming clinical test, gives reliable and reproducible results. It can be later modified to ventricular or lumbar infusion methods. It is possible only to measure R_{out} by this method.

By a ventricular drain inserted through a frontal burr hole 24 hours of ICP monitoring is done, baseline ICP obtained. The ventricular cannula is connected pressure monitoring system and after lumbar puncture the lumbar cannula is connected to an infusion pump. Infusion of ringer lactate starts at a rate of 0.5 -2 ml/ min and if necessary increased. The outlet of outflow tube is elevated in steps to increase the

ICP, and at each ICP level CSF outflow is measured. A minimum of 2 min is allowed between each measurement to obtain a steady state ICP on the new level. The surplus volume V_{out} is sampled from the outflow tube in periods of 3-5 minutes and measured at 6-7 different pressure levels.

A constant formation rate Fr of 0.4ml is presumed and CSF absorption V_{abs} is calculated

$$V_{abs} = V_{in} + 0.4 \quad V_{out}$$

Corresponding values of ICP and V_{abs} are plotted and the linear regression line is calculated by the method of least squares.

The bolus injection method

The bolus technique is fast and simple. A rapid intrathecal injection of a small volume – the bolus – results in an instant increase in ICP followed by a decline in pressure. The peak pressure should be greater than the baseline ICP and the rate of injection should exceed the formation rate. The rise in ICP depends on the compliance of the craniospinal space, and the subsequent decrease in ICP on both compliance and R_{out}. From the changes in pressure and the injected volume, R_{out} can be calculated by Marmarou's[1,2] formula.

Many authors agree that R_{out} value by bolus method is lower than that in perfusion, infusion methods. Even though bolus method is short some indeterminate part of the injected fluid is absorbed and does not contribute to the initial pressure rise.

Radio isotope dilution method

Radio isotope introduction, either by suboccipital or lumbar route into the ventricular system may depict the production, transport, and absorption of CSF. The tracer is followed by a gamma camera both in space and time over 1, 6 and 24 hours. Abnormalities in the scintigraphy can be described morphologically, (by ventricular dilatation or irregular or asymmetric cisterns) or dynamically (permanent or transient intra ventricular stasis, prolonged pericerebral transit time, half time greater than 12 hours and a transependymal efflux). The method has been widely used to illustrate the anatomic and dynamic conditions of the CSF compartment and to select patients with normal pressure hydrocephalus for CSF shunting.

Method	Advantage	Disadvantage
Constant pressure servo controlled method	More accurate	Time consuming and difficult
Constant infusion method	Faster than the other two infusion methods	The interpretation of the plateauing of the induced pressure rises, is difficult in plain infusion method
Constant infusion and constant pressure method	Open system makes it possible to avoid vascular reactions of raised ICP	Risk of infection
Bolus injection method (Marmarou)	Fast and simple	R_{out} value by bolus method is lower than that in infusion methods. Part of the injected fluid is absorbed and does not contribute to the initial pressure rise

CSF Dynamics studies in normal subjects

There are a few studies of CSF dynamics in normal subjects. The values of opening pressure in normal subjects in various studies are:

Borgesen and Gjerris (1987)[3] – 11.1 mmHg

Bono et al (2002)[4] -- 3.4 -12.2cms of H_2O[1]

The values of CSF outflow resistance in various studies are as follows:

Ekstedt et al[5] (1978) - 6.6 mmHg/ml/min,
Albeck et al[6] (1991) - 9.1 mmHg/ml/min,
Ramesh et al[7] (2005) – 5.98 mmHg/ml/min

The value of R_{out} increases with age.

CSF Dynamics in abnormal states

Normal pressure hydrocephalus

Aberrations in CSF flow dynamics generally are considered to be central to the development of normal pressure hydrocephalus (NPH). One proposed mechanism involves a transmantle pressure gradient wherein CSF pressure in the ventricles is greater than CSF pressure in the subarachnoid space. Short-lasting CSF pulsations (B waves) periodically apply pressure to the ventricular walls and have a water-hammer effect that enlarges the ventricles. Another mechanism involved in increasing transmantle pressure is impaired CSF flow, but the pressure gradient is not enough to raise intracranial pressure. Autopsy studies by Di Rocco et al[8] and Jellinger[9] has shown marked thickening of arachnoid in the basal cisterns in NPH.

The diagnosis of NPH is made solely on clinical symptoms in combination with interpretation of routine CT/MRI scans and not the response to shunt placement. Thus, a correct diagnosis of NPH can be associated with an unfavourable response to shunting, the symptoms may have progressed to a stage refractory to treatment. The terms shuntresponsive NPH and shunt-nonresponsive NPH are useful in clarifying the distinction between diagnosis and prognosis. The supplemental tests can be evaluated with respect to their ability to identify shunt responsive NPH.

From the studies by Petersen et al.[10] (1985), Vanneste et al.[11] (1993), Takeuchi et al[12](2000) it is clear that the prognostic value of CT/MRI scans is limited and that other supplementary tests are necessary to increase the prognostic accuracy of identifying NPH responders to shunting.

In the Dutch NPH study, Boon et al. [13] (1997) examined whether measurements of R_{out} predicted outcome after shunting for NPH patients, by the constant flow infusion method. This was a four-centre prospective study, and patients who fulfilled strict clinical criteria underwent shunt surgery irrespective of the R_{out} value. Values of R_{out} ranged from 6.3 to 42.3 mm Hg/ml/min, with an average of 17.3 mm Hg/ml/min.

Retrospectively, the authors recommended 18 mm Hg/ml/min as the Ro threshold on the basis of optimal sensitivity, specificity, Positive predictive value (PPV), and negative predictive value (NPV) values. At 18 mm Hg/ml/min, sensitivity and specificity equalled 46 and 87%, respectively. The calculated overall accuracy equalled 67%. The PPV and NPV equalled 92 and 34%, respectively. They concluded as measurement of R_{out} reliably predicts outcome if the limit for shunting is raised to 18 mm Hg/ml/min. At lower R_{out} values, the decision depends mainly on the extent to which CT and clinical findings are typical for NPH.

Takeuchi et al.[12](2000) used the bolus method for calculation of R_{out} in 25 shunted NPH patients in a retrospective study. The average R_{out} for the shunted group with improved outcome equalled 35.3 mm Hg/ml/min. and for the shunted group with no improvement equalled 9.1 mm Hg/ml/min. The calculated sensitivity and specificity equalled 100 and 92%, the overall accuracy equalled 96%. The R_{out} values were not used to select patients for shunting but rather were analyzed postoperatively to identify prognostic thresholds.

Meier and Bartels[14] studied 200 patients with suspected NPH. These were classified as “early” or “late” NPH by use of compliance computed from an infusion test. Improvement in symptoms was found in

65% of patients with early-stage NPH and 50% with advanced-stage NPH. The calculated PPV equalled 56%.

Kahlon et al[15]studied 68 patients with suspected NPH, 51 of whom had idiopathic NPH. Only patients with either a positive CSF tap test or a positive Katzman infusion test were shunted (R_{out} threshold, 14 mm Hg/ml/min).

	R_{out} threshold in mmHg	**PPV**
Boon et al., 1997	>18	92%
Børgesen et al.[16], 1979	>8	96%
Kahlon et al., 2002	>14	80%
Meier and Bartels, 2001	>13	13.56%
Gjerris et al.[17], (1982)	>12.5	

The other supplementary tests for diagnosing NPH and predicting the shunt response are:

CSF tap tests : In this test 30-50 ml CSF is drained and clinical improvement is looked for.

External Lumbar Drainage (ELD): Continuous drainage of CSF by lumbar catheter at the rate of 5-10ml/hr for 2 – 7 days is done and clinical improvement is looked for.

CSF flow void across the aqueduct in MRI scan due to hyperdynamic CSF circulation. (Bradley et al[18] 1986)

CSF aqueductal stroke volume > 42, by phase contrast MRI scan. (Scolatto et al.[19] 2008)

Expert opinion places the opening pressure range for NPH between 3 and 18 mm Hg (4–24.5 cms H2O).[20]

Evidence-based guidelines[21] for use in supplementary tests have been developed. A positive response to a 40- to 50-ml tap test has a higher degree of certainty for a favourable response to shunt placement but low sensitivity (26–61%). Determination of the CSF outflow resistance carries a higher sensitivity (57–100%) and a similar positive predictive value of 75 to 92%. Prolonged external lumbar drainage in excess of 300 ml is associated with high sensitivity (50–100%) and high positive predictive value (80–100%).

POST MENINGITIC HYDROCEPHALUS

Richard Winn et al.[22] (1979) in their study demonstrated that experimental bacterial meningitis in the rabbit induces marked alterations in the hydrodynamics of the CSF, as exemplified by an increase in the resistance to CSF outflow. This change was apparent in both pneumococcal and E. Coli meningitis and resolved slowly after

successful antibiotic therapy in the former. In contrast, steroid therapy in the acute stage of pneumococcal disease reduced CSF outflow resistance towards control levels.

Kemaloglu et al.[23](2002) noted that patients with TBM with mild and moderate hydrocephalus who underwent early shunt surgery (two days after diagnosis) had better outcomes compared to those who had delayed surgery (three weeks after diagnosis). This effect was not seen in patients with severe hydrocephalus. While this suggests that early surgery should be offered to patients with mild and moderate hydrocephalus without delaying it while waiting to determine the effect of medical therapy, it is unclear whether shunt surgery could have been avoided in some of these patients.

Rajsekhar et al. [24-25] (1991) have proposed the Vellore grading system for clinical grading of patients with TBM and hydrocephalus with grade I being the best grade and grade IV being the worst grade. The management of hydrocephalus can include medical therapy with dehydrating agents, steroids and anti tuberculous therapy for patients in good grades and those with communicating hydrocephalus. Surgery is required for patients with obstructive hydrocephalus and those in poor grades. However, the patients should be monitored carefully for any

worsening of sensorium or lack of improvement with medical management and a shunt might have to be done promptly. Surgery for patients in Vellore grade IV is usually associated with a poor outcome and high mortality and therefore recommended that these patients initially undergo an external ventricular drainage and only those patients who improved with external ventricular drainage be selected for surgery. They also found that clinical grade was the single most important factor in determining the final outcome.

Post traumatic hydrocephalus:

Marmarou et al[27] (1996) in their study on CSF dynamics in post traumatic hydrocephalus grouped patients with post traumatic ventriculomegaly based on ventricular size presence of atrophy , CSF dynamics measured by bolus method as

- Normal group
- Benign ICT group
- Atrophy group
- High pressure hydrocephalus group
- Normal pressure hydrocephalus group

Opening pressure above 15mmHg (20 cms of H_2O) and R more than 6mmHg/ml/min was considered elevated.

He suggested that, the diagnosis of post traumatic hydrocephalus (PTH) by serial CT scans alone is unreliable because it progressive ventricular dilatation, periventricular lucency were observed not only in post traumatic hydrocephalus but also in post traumatic atrophy.

The suggested procedure is

1. Measure the frontal horn index from CT scan, if it >0.3 CSF dynamics study should be performed by lumbar puncture.

2. A lumbar pressure ≥15 mmHg independent of R_{out} indicates a high pressure hydrocephalus and shunt placement is necessary.

3. A pressure < 15 mmHg with $R_{out} \geq 6$ mmHg/ml/min indicates a normal pressure hydrocephalus and shunt placement is recommended.

4. A normal pressure and R_{out} indicates a post traumatic ventriculomegaly secondary to atrophic process and patients are unlikely to improve from shunt placement.

Choi et al[28] have stated that extended craniectomy and repeat operation may play a role in the development of PTH in patients receiving decompressive craniectomy (DC) and future studies are required to ascertain CSF flow changes in patients who had DC by CSF dynamic study.

The mechanism of Post traumatic hydrocephalus following decompressive craniectomy described in literature are : Disturbance of pulsatile dynamics due to opening of cranial vault leads to decreased CSF outflow BIB, Mechanical blockage or inflammation of arachnoid granulations by post surgical debris.

Waziri et al[28] suggested that disruption of pulsatile ICP dynamics secondary to opening the cranial vault possibly results in decreased CSF outflow. If this is the case, early cranioplasty should lead to restoration of normal intracranial pressure dynamics and spontaneous resolution of hydrocephalus.

Foroglou[30] described obliteration of subarachnoid spaces with fibrous thickening of the leptomeninges, particularly in the sulci of the convexities and the base of the brain, in PTH and suggested that the obstruction around the convexities can result in hydrocephalus.

MATERIALS AND METHODS

The study was conducted at the Institute of Neurology, Madras Medical College and Government General Hospital, Chennai from June 2006 to January 2010.

Patients with communicating hydrocephalus admitted at the institute were included in the study. The patients were divided into three groups

- Normal pressure hydrocephalus
- Post traumatic hydrocephalus
- Post meningitic hydrocephalus

Normal pressure hydrocephalus group:

Patients with one or more of the triad of symptoms of gait abnormality, dementia and incontinence with CT scan or MRI scan brain showing dilated ventricles were included. Based on the NPH guidelines published in 2005[20], patients were sub classified as probable NPH and possible NPH. (Vide Appendix6) Patients satisfying the guidelines for unlikely INPH were excluded from this group

In all the patients, bifrontal index was calculated from CT or MRI scan which is calculated by measuring the maximal diameter of the frontal horns of lateral ventricle at the slice where frontal horns are largest to the width of the cranial cavity measured between the inner tables at the same level.

All the patients underwent cerebrospinal fluid opening pressure, pressure volume index and outflow resistance measurement by bolus lumbar injection method based on NPH guidelines 2005[21] which states 'to avoid complications and improve the certainty of a positive shunt response beyond 50 to 61%, all probable and possible INPH patients should be considered for supplemental testing.

All patients who had opening pressure in the range of 6-24 cms of water and outflow resistance above 18 mmHg were advised to undergo ventriculo peritoneal shunting.

The response to shunting was assessed before discharge from hospital and during follow up visits using assessment of gait and black shunt outcome scale (Vide Appendix 7). The follow up period ranges from 3 months to 36 months. The value of dynamics studies in the management and outcome was then analysed.

The second group consists of patients admitted with communicating hydrocephalus and with history of tuberculous meningitis in the recent past. For all of them P_o and R_{out} were measured on the day of admission. All patients with low GCS or papilloedema, or periventricular lucency associated with ventriculomegaly were advised shunt at the earliest (group 1). The patients without the above clinical /radiological evidence were observed with conservative management for 7-10 days from admission. Those who improved symptomatically and/or radiologically were continued with conservative management (group 3). Those who did not improve were advised shunt (group 2). The measurement of CSF dynamic studies of the above three groups were then compared.

The third group consists of patients with communicating hydrocephalus subsequent to head injury. CSF dynamics study was done for all the patients soon after the radiological evidence of ventriculomegaly was noted. Management was done based on the suggestion of Maramrou et al.[27] – all patients with elevated P_o ($\geq$ 15mmHg) and/or elevated R_{out} ($\geq$ 6mmHg) were shunted.

The method of dynamics study

Improvised bolus injection method using a bed side saline manometer devised at the Institute of Neurology, Chennai – the Madras Institute of Neurology (the MIN) method was used.

The apparatus includes a saline stand, one meter scale, intravenous set, 3-way adapter, and a 20G lumbar puncture needle. The scale is mounted on the saline stand and intravenous set is mounted over it. The intravenous set is filled with saline up to 11cms of water with zero level corresponding to the spine of the patient who is in lateral position. Then lumbar puncture is performed with 20G spinal needle and, the needle is connected to the saline manometer through the 3-way adapter without letting out any CSF. After the saline column stabilises the opening pressure P_o is noted. A known volume of saline (rV), usually 5-10 ml is injected into the subarachnoid space through the 3-way port at the rate of 1ml/second. The peak pressure (P_p) reached after the bolus injection is noted. The saline column in the manometer starts falling gradually. After a certain time (t in minutes) the pressure recording in the manometer (P_t) is noted. The CSF outflow resistance is calculated using two steps formula described by Marmarou:

First step : pressure volume index

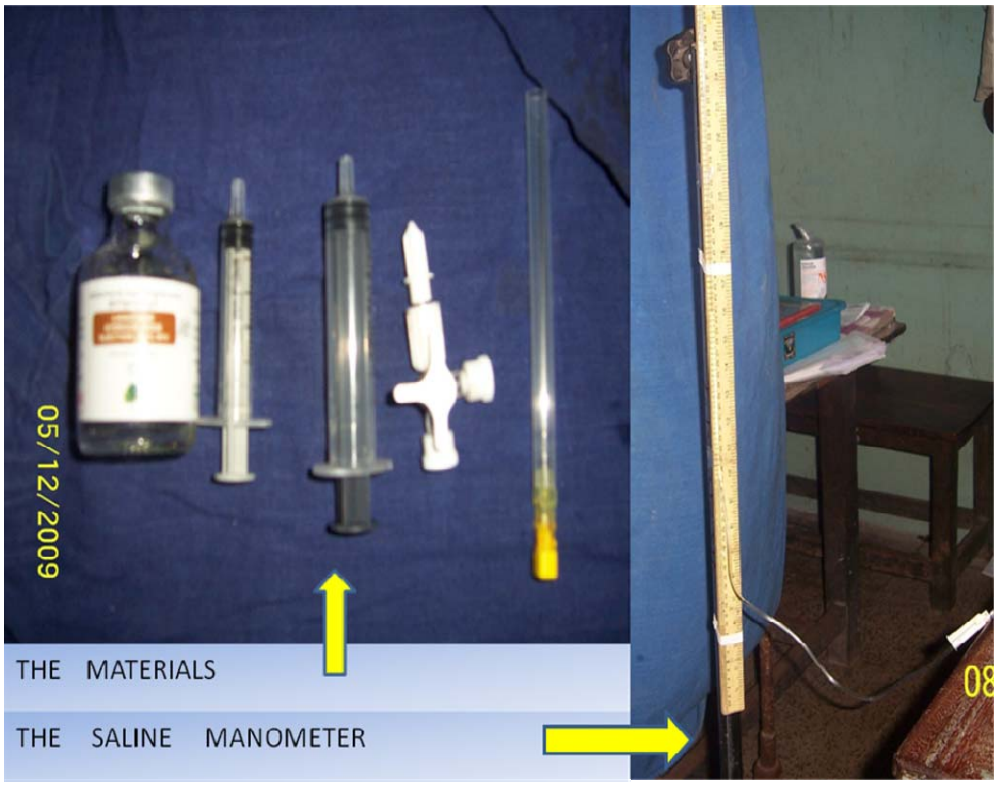
$$PVI = \frac{rV}{\log\left(P_p / P_o\right)}$$

Second step: outflow resistance

$$R_{out} = \frac{tP_0}{PVI\left[\log\frac{P_t(P_p - P_o)}{P_p(P_t - P_o)}\right]}$$ cms of H_2O /ml/min

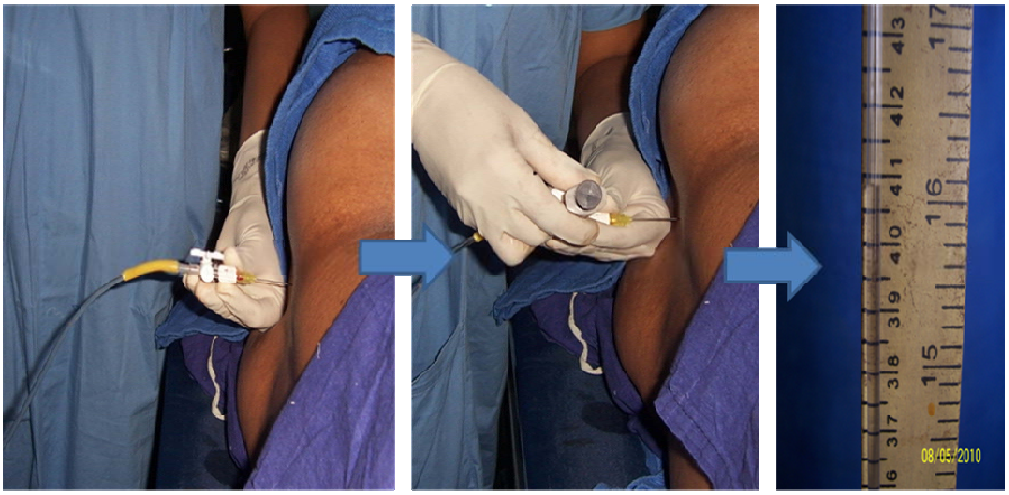
This is converted into mm.Hg/ml/min (divided by 1.36).

MATERIALS AND METHODS



THE MATERIALS

THE SALINE MANOMETER



Lumbar puncture, connected to manometer

The bolus of saline being injected

The pressure reading

RESULTS

NORMAL PRESSURE HYDROCEPHALUS:

The first group of patients is that of normal pressure hydrocephalus.

NORMAL PRESSURE HYDROCEPHALUS								
s. no	age	sex	clinico radiological diagnosis	DYNAMICS			management	black shunt outcome
				Po cms of H2O	PVI	Rout mmHg		
1	45	M	probable NPH	17	27.13	21.97	shunt	excellent
2	59	M	probable NPH	20	27.32	65.35	shunt	excellent
3	65	M	probable NPH	12	51.6	4.12	conservative	NA
4	60	M	probable NPH	20	18.88	55.82	shunt	dead
5	75	F	possible NPH	11	34.5	6.4	conservative	NA
6	60	M	possible NPH	12	35.56	6.614	conservative	NA
7	85	M	possible NPH	11	33.6	6.2	shunt	dead
8	72	M	possible NPH	11	35.2	7.1	conservative	NA
9	68	M	possible NPH	11	35.37	6.8	conservative	NA
10	77	F	possible NPH	17	41	28.09	not willing for shunt	NA
11	74	M	possible NPH	11	36.2	6.6	conservative	NA
12	53	F	possible NPH	11	36.4	6.16	conservative	NA
13	63	M	probable NPH	14	24.3	25.33	not willing for shunt	NA
14	70	F	probable NPH	11	30.73	19.1	not willing for shunt	NA
15	70	M	possible NPH	12	28.4	19.8	shunt	excellent
16	65	M	probable NPH	14	26.2	24.9	shunt	excellent
17	68	F	probable NPH	11	29.3	21.4	shunt	excellent
18	60	F	probable NPH	14	32.6	18.6	shunt	good
19	64	M	probable NPH	13	25.8	23.3	shunt	good

(NA – Not Applicable)

NPH – AGE and SEX DISTRIBUTION

Age	No.	%
< 50	1	5.3
50 - 60	5	26.3
60 - 70	8	42.1
> 70	5	26.3
Total	19	

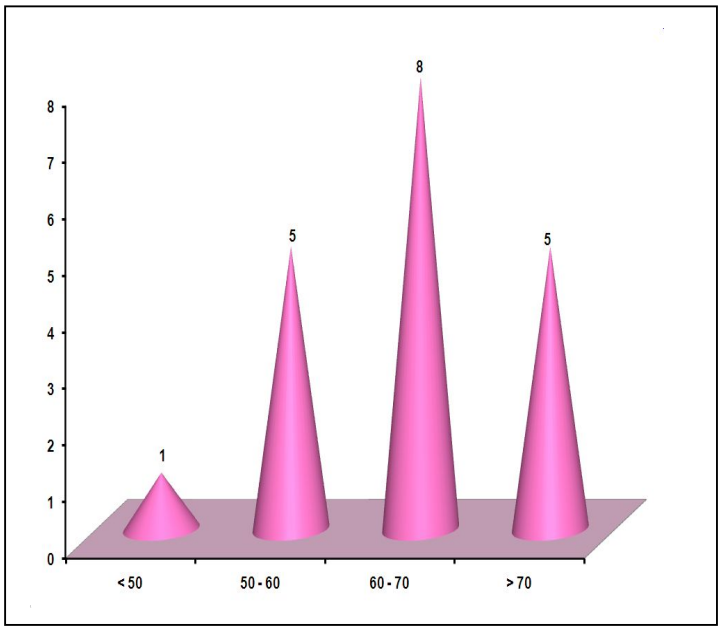


Figure 1: NPH AGE DISTRIBUTION

SEX	No.	%
Female	6	31.6%
Male	13	68.4%

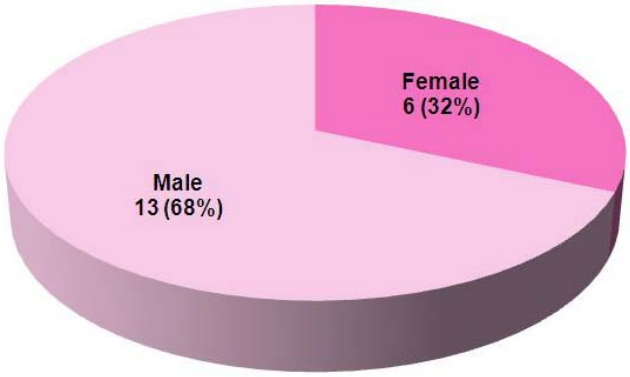


Figure 2: NPH - SEX DITRIBURION

Clinico– radiological classification of the cases:

Out of the 19 patients included in the group, 10 patients were sub classified as 'Probable NPH' and 9 as 'Possible NPH', based on the criteria of NPH guidelines published in 2005.[20] Among the 9 patients with diagnosis of possible NPH, 5 had co-existing other neurological illness like cerebrovascular accident with infarct, Parkinsonism, etc. 1 patient had a sub acute onset of symptoms. Radiologically, 8 of them had atrophic brain changes, thus these factors confounding the definitive diagnosis of NPH clinico radiologically alone.

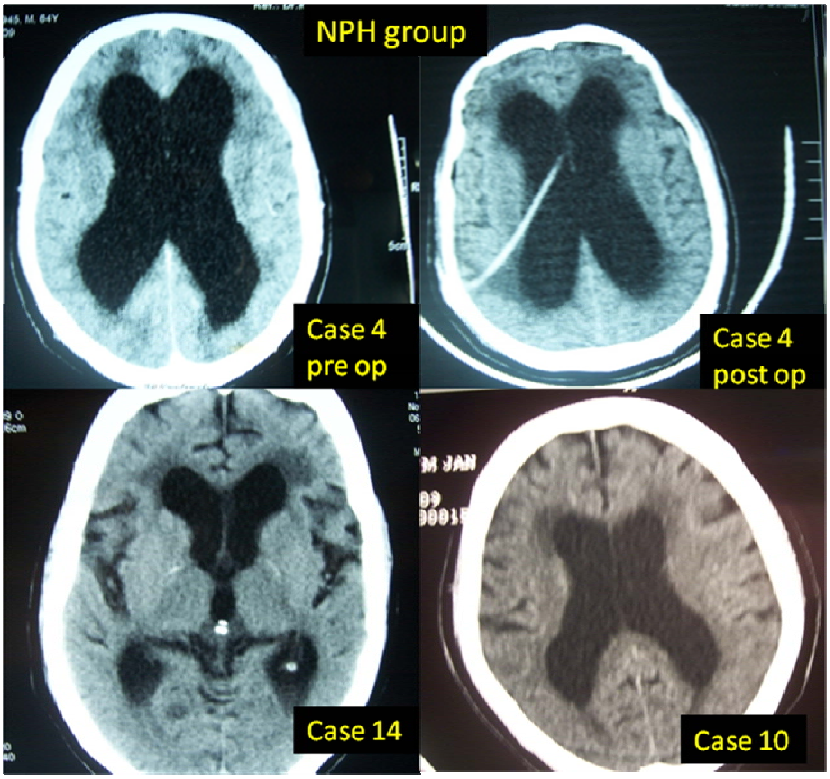


Figure 3: CT Scan –NPH group

Po and R_{out}:

In all the 19 patients opening pressure was between 11 and 20 cms of H_2O, which is within the normal range for NPH (6-24 cms of H_2O).[20]

11 patients out of 19 had R_{out} more than 18 (range 18.6 to 65.35) and 8 patients had 6.6 or less. There were no patients who had R_{out} values in the intermediate range of 7 – 18.

(The threshold value for raised R_{out} for NPH coated in various studies ranges from 8 to 18 mmHg/ml/min)

Clinico-Dynamic co-relation:

Out of the 11 patients with increased R_{out} 9 were from probable NPH category and 2 from possible NPH category. In other words, of the total 10 patients with probable NPH, 9 had raised R_o and among 9 patients with possible NPH 2 had raised R_{out}.

Out of the total 19, 8 patients had normal R_{out} and Po. 7 of them were from possible NPH category and 1 was from probable NPH category.

	Probable NPH	Possible NPH	
R_{out} Increased	9	2	11
R_{out} Not increased	1	7	8
	10	9	19

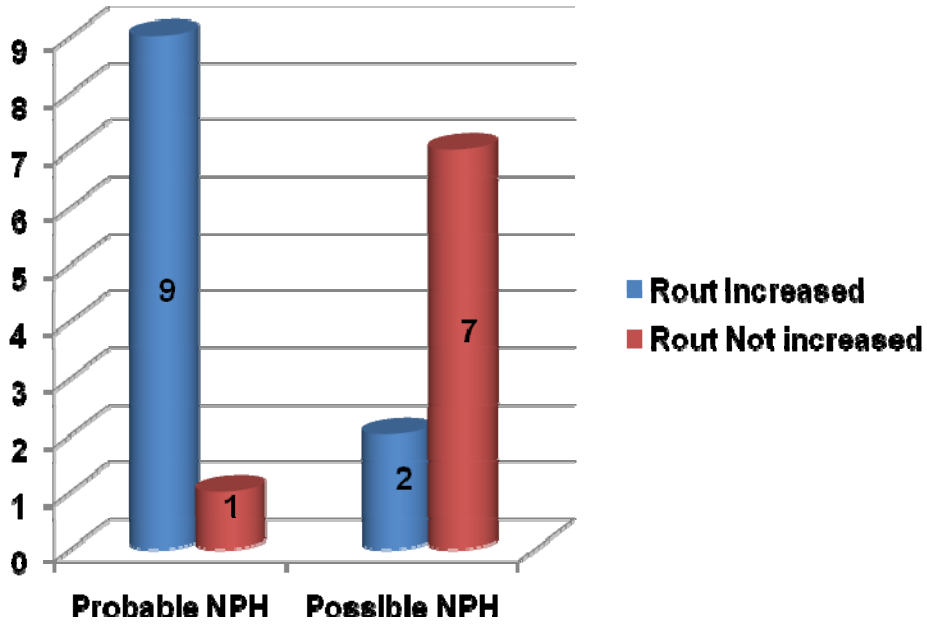


Figure 4 : clinico - dynamic co-relation

This is indicating **a good clinico–dynamic co relation and the complementary value of CSF dynamic studies in the diagnosis of NPH**.

Shunt Responsiveness:

	Predicted Responder		Predicted Non responder	
	Shunted	Shunt not done	Shunted	Shunt not done
Improved	36.8	0	0	10.5
Not improved	5.3	15.8	5.3	26.3

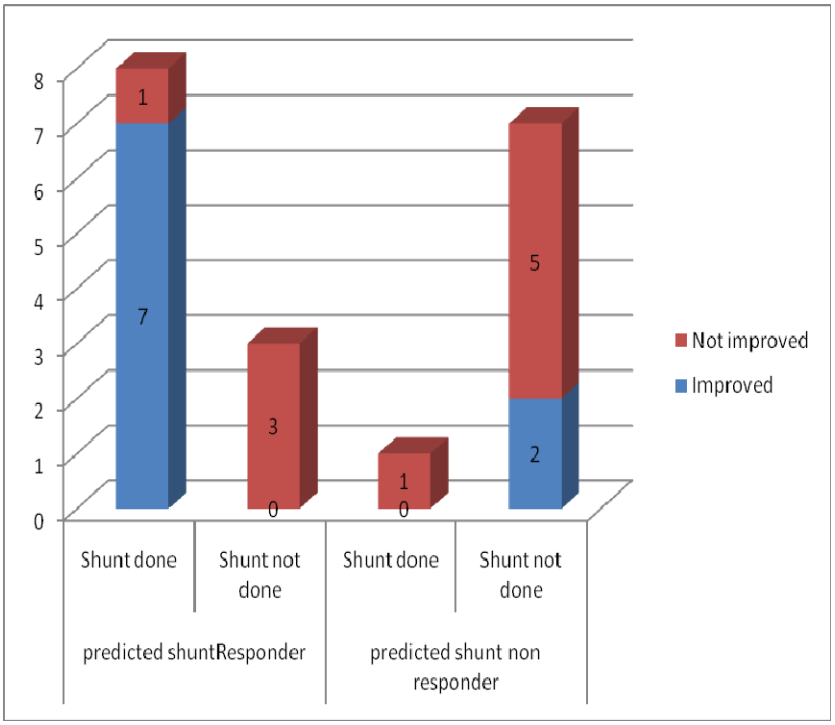


Figure 4

11 patients out of 19 had R_{out} more than 18 and hence were expected be shunt responsive.8 patients out of the 11 underwent ventriculo peritoneal shunt. Outcome was assessed by Black shunt outcome scale (vide appendix 7 **).**

7 patients out of the 8 who underwent shunt had excellent/ good outcome and one patient expired 25 days after shunt due to myocardial infarction.

Out of the total 19, 8 patients had normal R_{out} and Po. They were all hence expected to be shunt non-responders.

Among the 8 patients with normal R_{out} 7 were managed conservatively without shunt and 2 of them showed some improvement in their symptoms and 5 remained static.

7 patients out of 8 had atrophic changes brain which could explain the dilated ventricles and 4 out of 8 had other neurological problems that could partly explain the symptoms and signs.

1 patient with normal R_{out} underwent ventriculo peritoneal shunt because he had periventricular lucency. He also had multiple co morbid factors and old cerebrovascular accident and expired post operatively.

Shunt Vs Outcome

Out of total 19 patients 9 patients underwent ventriculo peritoneal shunting. 8 of them had been predicted to be shunt responders by dynamics, and 1 as shunt non responder. Of the 8 predicted Shunt Responsive NPH, 7 had good/excellent outcome and 1 patient expired.

The one patient who was predicted to be non-responder and underwent shunt expired. 3 patients, who were predicted to be Shunt Responsive NPH but refused shunt, remained static.

	Shunted and Good outcome	**Shunted and Poor outcome**
Predicted as shunt responsive	7	1
Predicted as shunt non responsive	0	1

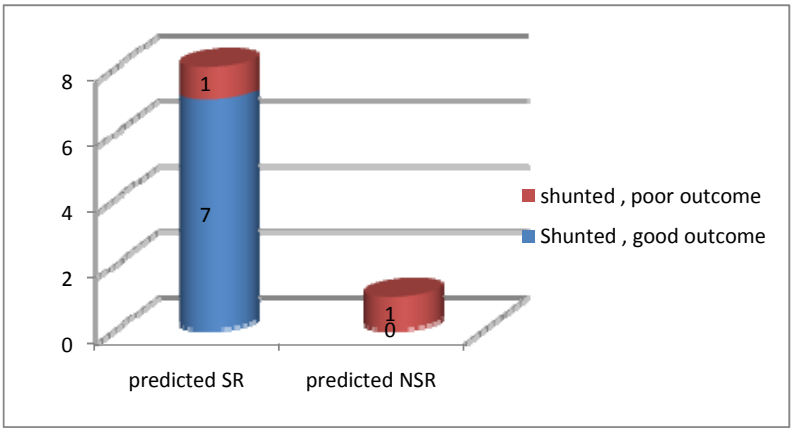


Figure 5 : Shunt Vs Outcome (3 patients out of 11 with raised R_{out} were not willing for shunt and remained static, hence excluded from the above table)

The prediction of shunt responsiveness by cerebrospinal fluid dynamics studies correlates with good outcome in 87.5% (7/8 cases).

In the above chart we can see that 87.5% of the patients predicted as shunt responders had good outcome after ventriculo peritoneal

shunting and one patient who was predicted as shunt non responder by dynamics and underwent shunt did not improve and expired.

Statistical Analysis

Hypothesis: Prediction of shunt responsiveness by dynamics study co relates with outcome.

Test: One-Sample Kolmogorov-Smirnov Test

One-Sample Kolmogorov-Smirnov Test	
N	9
Kolmogorov-Smirnov Z	1.412
Asymp. Sig. (2-tailed)	.037

a Test distribution is Normal.

b Calculated from data.

P-Value – 0.037 < 0.05. This is statistically significant.

Inference: **Prediction of shunt responsiveness by dynamics study co- relates significantly with outcome**.

POST MENINGITIC HYDROCEPHALUS:

The second group consists of 23 patients with communicating hydrocephalus and history of meningitis in the recent past.

s. no	age	sex	Papillodema	PVL	OP cms of H2O	PVI	Rout mmHg	Mx	outcome
1	18	M	no	no	14.5	27.5	7.95	cons	improved
2	16	F	no	no	24	19.1	60	shunt	improved
3	38	F	no	no	11	69	6.5	cons	improved
4	37	M	present	present	72	49.12	4.556	shunt	expired on 5th POD
5	13	F	present	no	33	23.386	30	shunt	improved
6	22	F	no	no	30	12.57	11.35	shunt	improved
7	50	M	no	present	34.5	52.3	5.92	shunt	improved
8	45	M	no	no	24	17.86	13.94	shunt	improved
9	45	M	no	no	11	31.2	6.4	cons	improved
10	40	M	no	present	21	43	8.27	shunt	improved
11	33	F	no	present	11	26.46	28.9	shunt	improved
12	18	F	present	present	28	21.5	44.46	shunt planned	expired
13	30	M	no	no	12.5	22.22	28.34	shunt	improved
14	33	M	no	no	45	22.32	25.31	shunt	improved
15	17	F	present	present	17.5	17.76	122	shunt	improved
16	52	M	no	no	46	23.55	11.5	shunt	expired
17	48	M	no	present	31	18.9	49.28	shunt	Improved
18	33	M	present	no	25	22.52	25.44	shunt	improved
19	36	F	no	present	39	23.1	26.33	shunt	improved
20	23	F	present	present	31	23.56	24.12	Shunt	improved
21	33	M	no	present	28	22.38	28.4	Shunt	improved
22	37	M	present	no	29	24.4	23.5	Shunt	improved
23	21	M	no	no	26	20.6	26.1	Shunt	improved

Of them 14 were females and 9 were males. Their age ranged from 13 years to 52 years.

AGE DISTRIBUTION

Age	No	%
< 20	5	21.7%
20 - 40	13	56.5%
> 40	5	21.7%

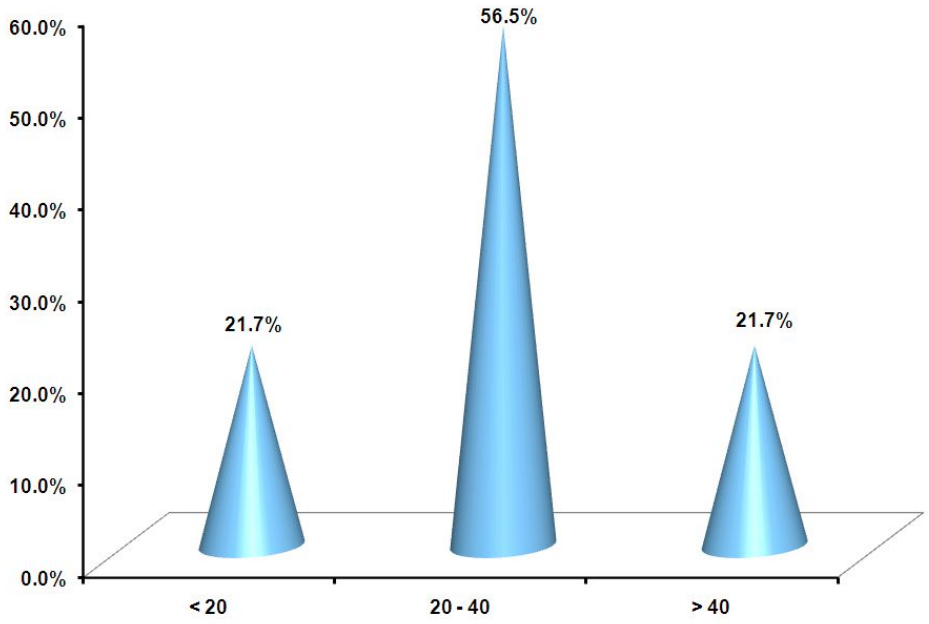


Figure 6: Post meningitic Hydrocephalus - Age distribution

SEX DISTRIBUTION

Sex	No	%
Female	9	39.1%
Male	14	60.9%

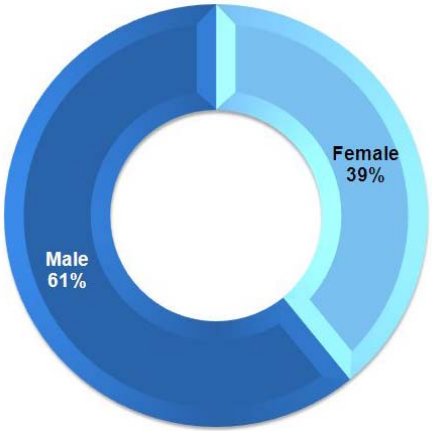


Figure 7: Post meninigitic Hydrocephalus - sex distribution

Clinical Features:

Clinically 7 of them had established papilloedema, with associated 6th nerve palsy in 4 and low Glasgow Coma Scale (GCS) in 2. 1 patient had 6th nerve palsy with secondary optic atrophy. 3 patients had other ocular findings like 3rd nerve palsy, tubular field. 2 of them had multiple cranial nerve palsy due to basal archnoiditis. Others had headache, vomiting or drowsiness.

Radiology: Radiologically in CT scan brain all the patients had bifrontal index more than 0.3 (range 0.33 -0.55). Periventricular lucency was present in 10 cases and cortical sulci were effaced in 18 cases.

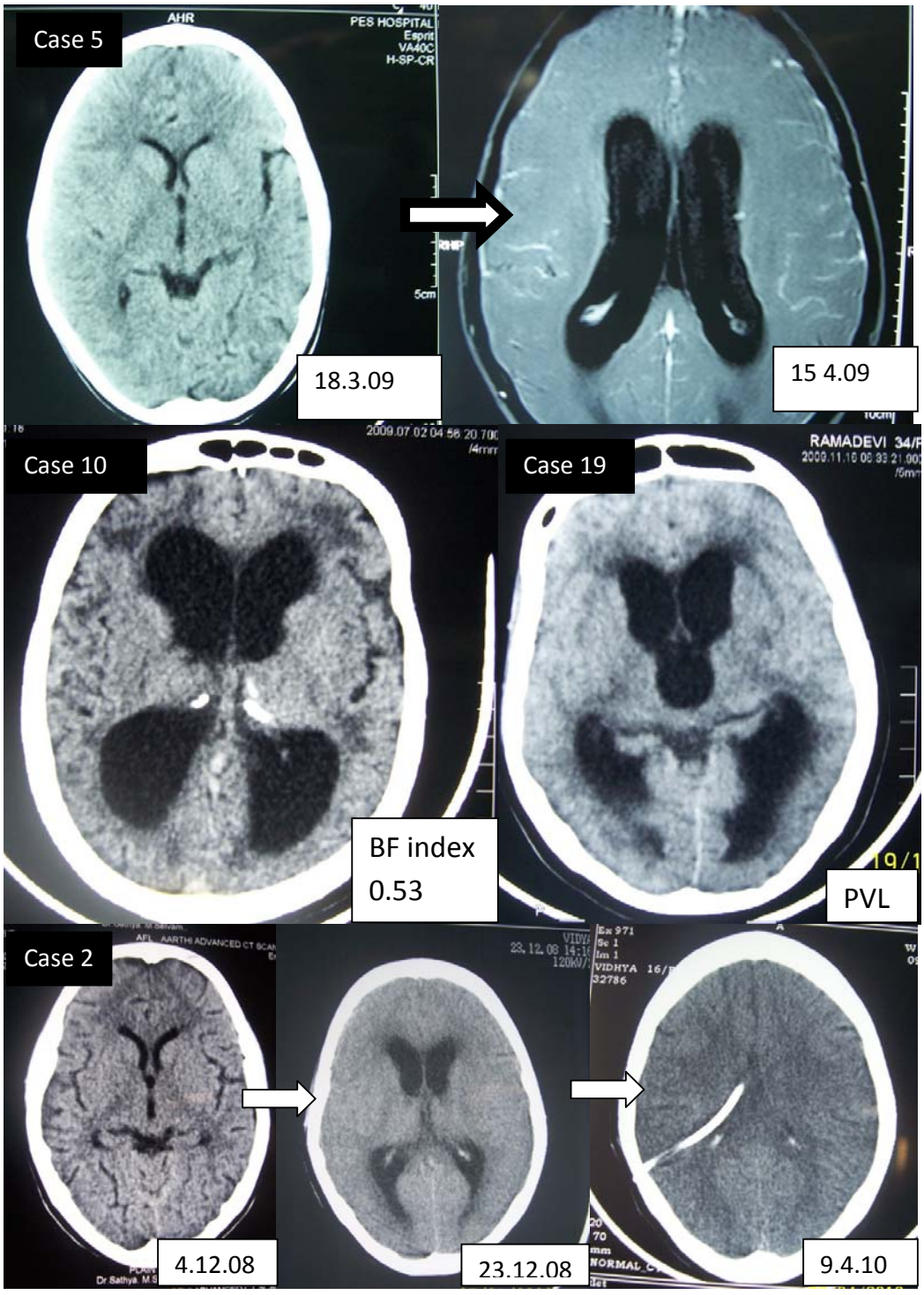


Figure 8 : CT Brain - Post meningitic hydrocephalus

Management

Out of 23 patients, papillodema and/or periventricular lucency were present in 13. One of them expired on the day of admission before further management. The remaining 12 patients underwent ventriculoperitoneal shunting at the earliest (group 1).

10 out of 13 had neither papilloedema nor periventricular lucency and were in GCS 14 or 15. They were managed with medically and with close observation for a period of 7-10 days.

7 of them who remained static or deteriorated clinically/ radiologically during the waiting period underwent ventriculoperitoneal shunting (group 2).

3 patients improved clinically/ radiologically with conservative management and did not undergo shunt (group 3). In these 3 patients, 2 had bifrontal index of 0.36 with cortical sulci seen and no PVL, and improved with conservative management. The third patient was drowsy, with normal fundus, with bifrontal index of 0.38, effaced cortical sulci without PVL. She improved and became fully conscious and bifrontal index in follow up scan done was 0.34.

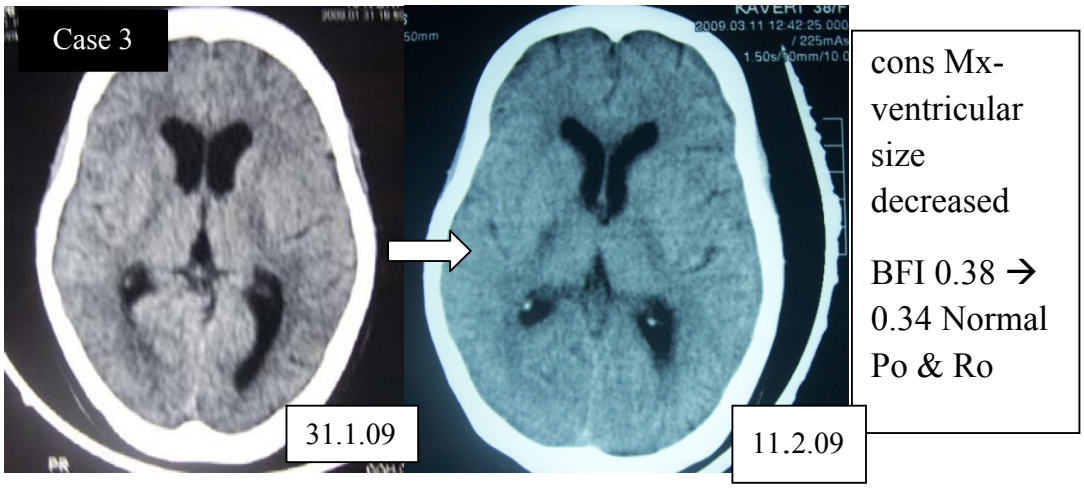


Figure 9

Analysis:

The dynamics studies in the three groups were compared.

	P_o range cms of H2O	**Mean Po ± STDEV**	**R_{out} range** mmHg	**Mean Ro ± STDEV**
Group 1	11 - 72	30.76±14	4.5 - 122	32.4±30
Group 2	12.5 - 46	29.64±12	11.35 - 61	25.36±17
Group 3	11 - 14.5	12.17±2	6.4 - 7.95	6.95±0.86

From the above table it is evident that **the mean P_o and R_{out} in group 1 and 2 which required shunt is higher than the group which did not require a shunt.**

Taking the highest value of P_o and R_{out} in group 3 as threshold P_o >15cms of H2O and R_{out} >8mmHg/ml/min were considered raised and further analysis of three groups done.

	No. of cases			
	Total	**Raised P_o± raised R_{out}**	**Normal P_o, raised R_{out}**	**Normal P_o, R_{out}**
Group 1	13	12	1	0
Group 2	7	6	1	0
Group 3	3	0	0	3

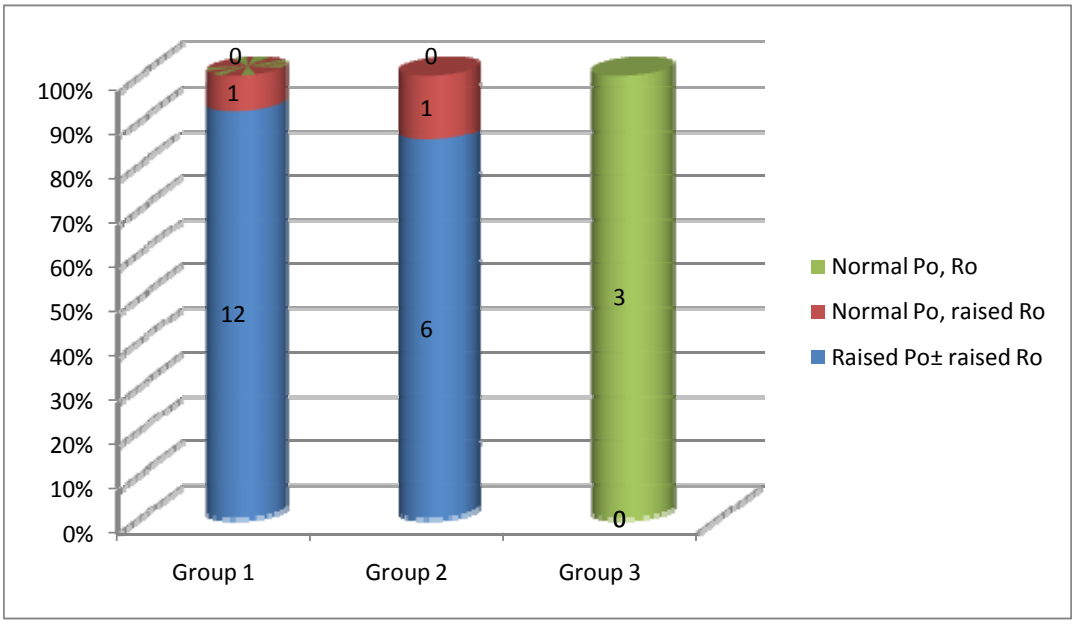


Figure 10: group Vs dynamics

In group 1 and 2 there was no patient who had normal P_o and normal R_{out}. 12out of 13 patients in group 1 had raised P_o with 10 of them having raised R_{out} also, and 6 out of 7 patients in group 2 had raised P_o and R_{out} High pressure hydrocephalus (HPH). One patient each in group 1 and 2 had normal P_o with raised R_{out} (NPH like).

Hence if **P_o > 15cms of H2O and R_{out} > 8 mmHg /ml/min were kept as threshold and as indication for surgery at admission** all patients in group 2 would have been shunted earlier.

Shunt Vs Outcome

Out of 20 patients in group 1 and 2, 19 patients underwent ventriculo peritoneal shunting. Among them 17 patients improved (89.5%) and 2 patients expired due progressive disease process with basal meningitis and multiple cranial nerve involvement.

1 patient out of 20 expired on the day of admission itself, before shunt could be done.

	improved	not improved
shunt done	17	2
shunt not done	3	0

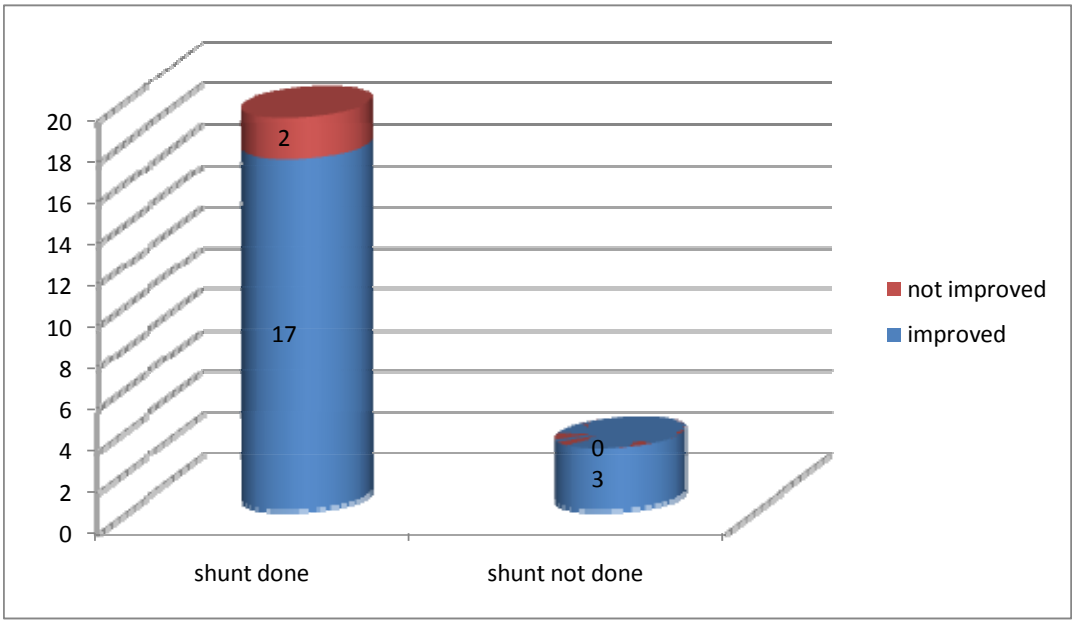


Figure 11 : Shunt Vs Outcome

Hence **the value of dynamics studies in predicting patients who need shunt is (89.5%) 17/19** and the value in predicting those who do not require shunt is (100%) 3/3.

Statistical Analysis

Hypothesis: Raised R_{out} and/or P_o is a good criteria for decision making regarding shunt.

Test – One sample t-test

One Sample t – test

	Test Value = 0					
					95% Confidence Interval of the Difference	
	t	df	Sig. (2-tailed)	Mean Difference	Lower	Upper
VAR0001	17.390	21	.000	1.0909	.9604	1.2214

P-value: $0.000 < 0.05$

This is highly statistically significant.

Inference: **Raised R_{out} and/or P_o is a good criteria for decision making regarding shunt.**

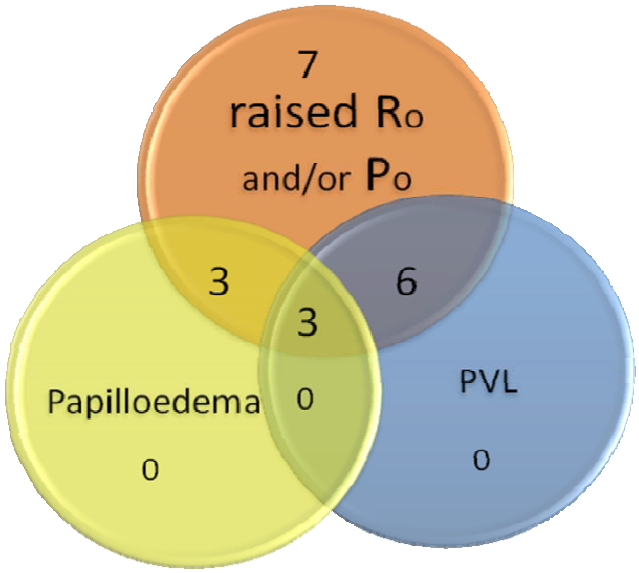


Figure 12

Out of the 19 patients who underwent ventriculo peritoneal shunting 3 patients had both papillodema and periventricular lucency, 3 had papillodema alone and 6 had periventricular lucency alone. There were 7 patients who had altered dynamics but had neither papilloedema nor periventricular lucency who also improved with shunt.

Hence **dynamics studies can detect patients who need cerebrospinal fluid shunting procedures earlier than clinical or radiological indicators** like papilloedema or periventricular lucency.

POST TRAUMATIC HYDROCEPHALUS

The third group consists of eight patients with post traumatic ventriculomegaly:

S. No	Age	Sex	OP cms of H2O	PVI	Rout mmHg	classification	Mx	clinical status	outcome GOS
1	20	M	24	22.42	24.4	HPH	shunt	improved	5
2	60	M	14	42.01	2.28	atrophy	cons	improved	4
3	55	M	14	26.8	5.55	atrophy	cons	static	3
4	45	M	11	33.56	2.23	atrophy	cons	static	2
5	34	M	11	20.11	27.8	NPH	shunt	improved	5
6	70	F	11	35.7	5.7	atrophy	cons	improved	5
7	30	M	39	23.58	15.9	HPH	Shunt	improved	4
8	47	M	11	34.2	5.57	atrophy	cons	Expired	1

The duration at presentation from trauma ranged from 15 days to 8 months. The earlier CT scan findings of head injury in these patients were subarachnoid haemorrhage, contusions brain, diffuse cerebral edema or subdural hematoma. They either got readmitted with fresh symptoms or

were found to have ventriculomegaly in follow up scan done during the first admission itself.

Age Distribution

Age	No	%
< 20	1	12.5%
20 - 40	2	25.0%
> 40	5	62.5%

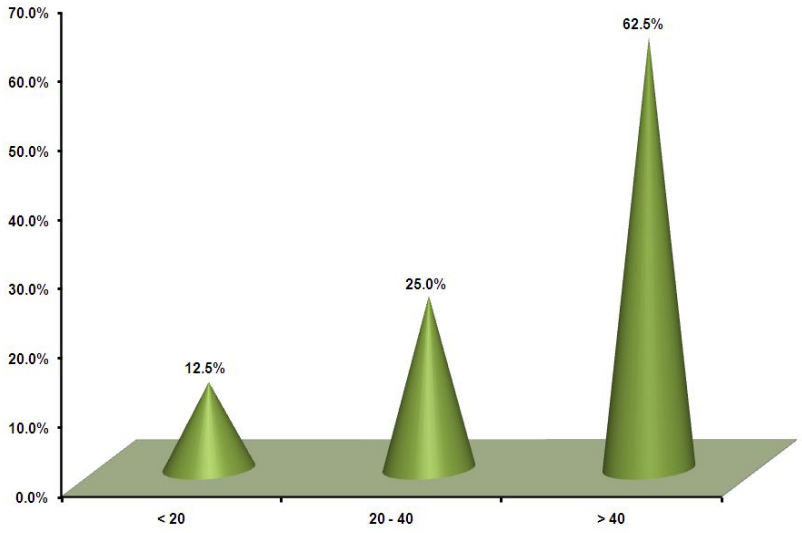


Figure 13: post trauma – age distribution

Sex Distribution

Sex Distribution	No	%
Female	1	12.5%
Male	7	87.5%

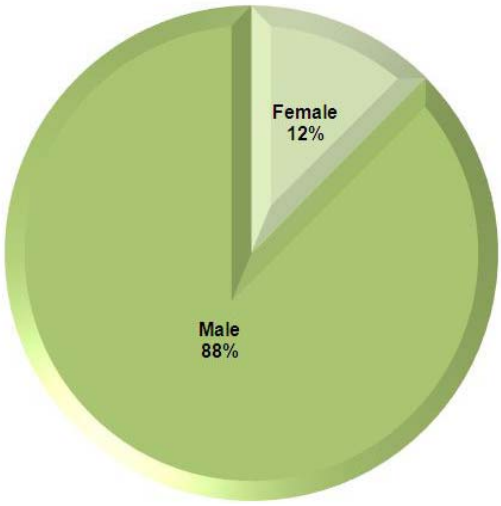


Figure 14 : post trauma – sex distribution

In all the patients, the bifrontal index in CT scan brain was more than 0.3, ranging from 0.35 to 0.44. Periventricular lucency was present in one patient and cortical sulci were effaced in another patient.

By lumbar bolus injection method opening pressure (P_o) and outflow resistance (R_{out}) measurement was done for all eight patients. The value of P_o more than 20 cms of H_2O and R_{out} more than 6mmHg/ml/min was considered elevated.

2 patients out of 8 were found to have elevated P_o and R_{out} suggestive of high pressure hydrocephalus and 1 patient was found to have normal P_o and elevated R_{out} suggestive of normal pressure hydrocephalus. All the 3 patients underwent ventriculo peritoneal shunt and improved after shunt with Glasgow Outcome Scale (GOS) 5 (Vide : Appendix 7).

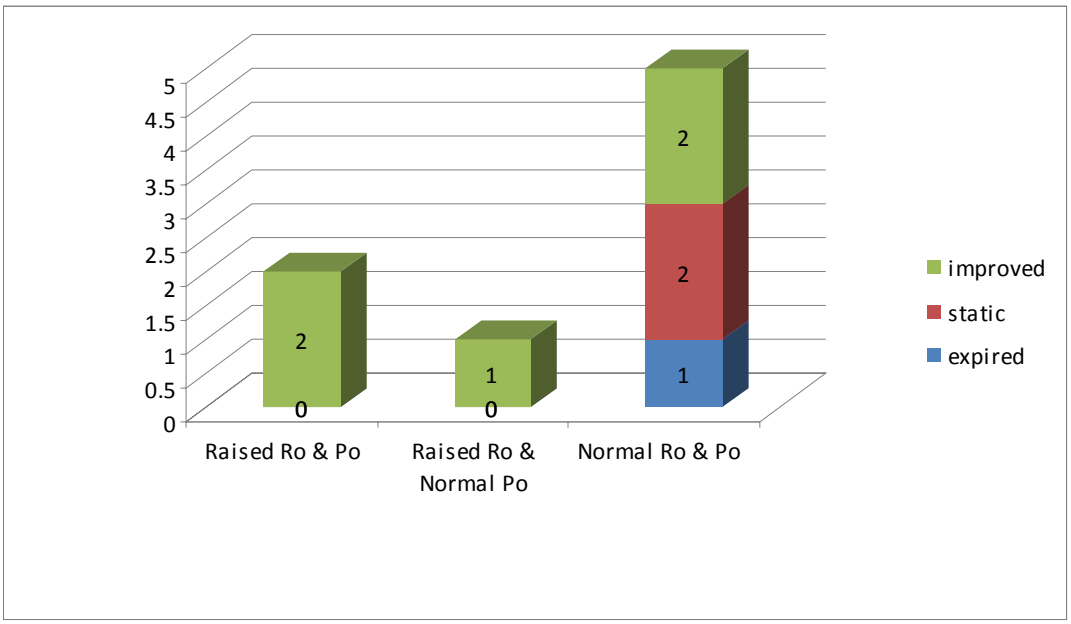


Figure 15 : Dynamics Vs Outcome

5 patients out of 8 had normal P_o and R_{out} suggestive of atrophic ventricular dilatation and did not undergo shunt. They were managed conservatively. Out of five, 2 patients improved with GOS 4, 2 remained static with GOS 2 and 3, one expired, GOS 1.

Dynamics Vs Etiology

Of the 3 patients who had hydrocephalus by dynamics studies, 2 had posttraumatic sub arachnoid haemorrhage and 1 had undergone decompressive craniectomy for sub dural hematoma. Whereas patients with atrophic ventriculomegaly by dynamics had multiple contusions brain and diffuse brain edema in immediate post trauma scans.

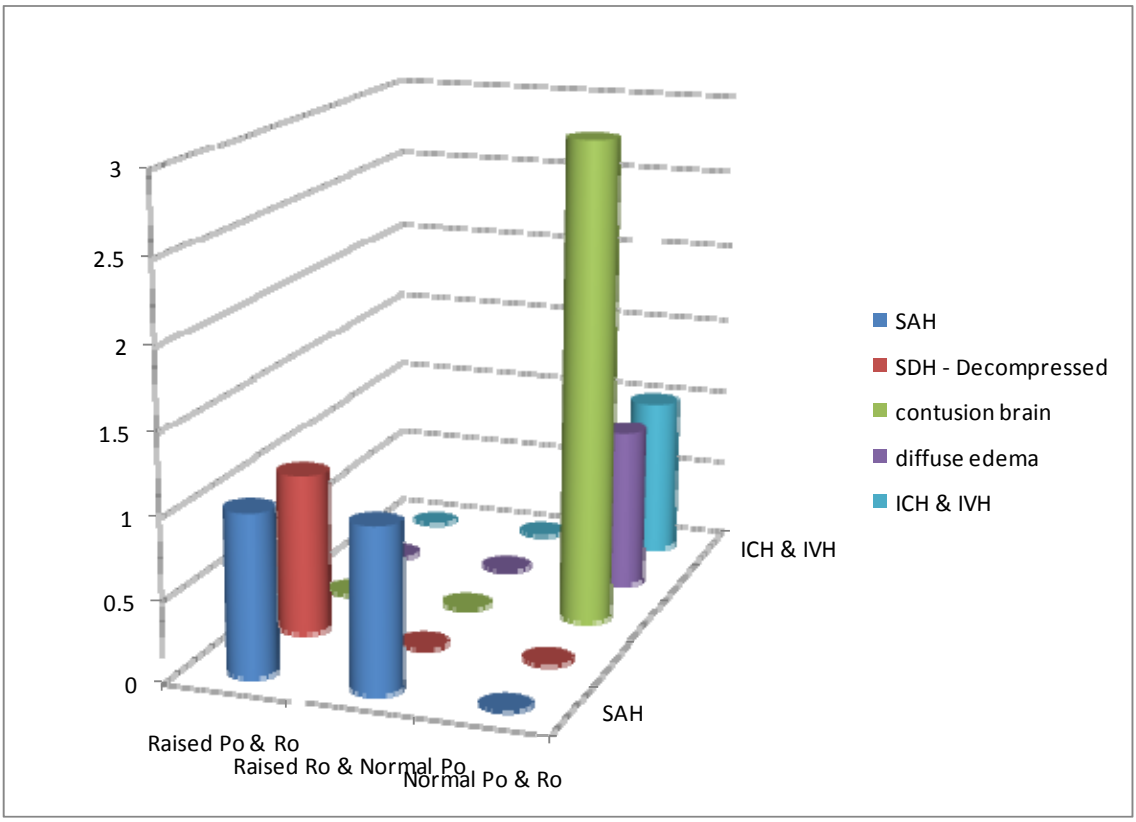


Figure 16 : Dynamics Vs Etiology

The average bifrontal index in the hydrocephalic group was 0.397, as against 0.37 in the atrophic group.

DISCUSSION

Disturbance in the cerebrospinal fluid flow at the basal cisterns or arachnoid villi is the basic pathology in communicating hydrocephalus. The various causes of communicating hydrocephalus are basal meningitis, traumatic brain injury, subarachnoid haemorrhage or idiopathic (NPH).

Subsequent to such disturbances in CSF flow there is immediate increase in outflow resistance and raise in ICP follows at various rates due to dilatation of ventricular system.

Often in such cases there is a diagnostic dilemma between hydrocephalus and atrophic dilatation of ventricles. This is often encountered in elderly patients with NPH or in patients with post traumatic cerebral atrophy. There are other clinical confounding factors hindering definitive diagnosis like other neurological illness in the elderly or neurological deficits due to basal arachnoiditis or traumatic brain injury. The clinician often finds it difficult to decide whether the present clinical condition is due to ventriculomegaly or other neurological deficits.

The next challenge would be to find out, which are patients who would respond to shunt surgery. In NPH the diagnostic criteria and shunt responsiveness are independent and a case of probable NPH may have irreverisible neuronal damage and may not respond to shunt.

There have been various studies attempting to find solution for the aforesaid challenges:

- The diagnostic dilemma
- Prediction of shunt responsiveness and choosing the correct patient for CSF diversion procedure.

One of the valuable tools which come to help in the above said challenges is study of cerebrospinal fluid dynamics by any of the established methods.

In this study, the CSF dynamics of 50 patients with communicating hydrocephalus – normal pressure hydrocephalus, post traumatic hydrocephalus, post meningitic hydrocephalus groups, were studied.

NORMAL PRESSURE HYDROCEPHALUS GROUP

There were 19 patients in this group and the opening pressure was between 11-20 cms of H_2O (10-14.7mmHg) with mean of 13.3 cms of

H_2O (9.8 mmHg) this is within the range of 2-20 mmHg coated by Marmarou et al[31]. and 4- 17.6 mmHg given in NPH guidelines 2005[20].

The threshold value of R_{out} for predicting shunt responsiveness quoted in various studies ranges between 8-18 mmHg/ml/min. In our study group, out of 19 patients, 11 had R_{out} values ≥ 18 mmHg/ml/min and were classified as predicted shunt responders. The remaining 9 patients had R_{out} < 7 mmHg/ml/min, and were classified as predicted non-responders to shunt. There were no patients in R range of 8-18 mmHg/ml/min (equivocal values).

It was found that **the dynamics studies co-related well with the clinico- radiological classification as probable and possible NPH**, with majority (9/10) of patients with probable NPH having elevated R_{out} and majority (7/9) patients with possible NPH having normal R_{out}.

In our study it was also found that **the prediction of shunt responsiveness by CSF dynamics co-relates with good outcome in 87.5% of cases**. This co-relates with the positive predictive value of R_{out} quoted in various studies, 92% by Boon et al.[13], 96% by Borgesen et al.[17]and 80% by Kahlon et al.[15]

By One-Sample Kolmogorov-Smirnov test we found that the value of CSF dynamics in predicting shunt responsiveness is statistically significant (P value – 0.037)

POST MENINGITIC HYDROCEPHALUS:

One of the most common complications of meningitis especially tuberculous, is hydrocephalus of the communicating type, which is due to the formation of thick gelatinous basal exudates around the interpeduncular and pontine cisterns in the acute stages and adhesive leptomeningitis in the chronic stages. However, in many cases, diversion of ventricular CSF through a VP shunt does not result in a significant improvement in their condition and many die despite the intervention. Therefore the possibility of factors other than hydrocephalus—for instance, arteritis, ischemia, and encephalopathy—being the cause of the altered sensorium and adverse neurological condition is to be considered. Thus selecting patients who would benefit from a CSF diversion procedure becomes important.

There have been attempts to form criteria for predicting shunt responsiveness, by clinical status of the patient (Rajasekar et al[26])

In this study, the CSF dynamics of post meningitic hydrocephalus patients were studied and its usefulness evaluated. This is the first study analysing CSF dynamics in post meningitic hydrocephalus patients in the available literature.

The mean P_o and R_{out} values of patients who underwent early or delayed shunt (group 1&2) were found to be higher than the group who did not require a shunt (group3). Hence a threshold value of P_o and R_{out} above that of highest value of group 3 would help to select patients for shunt by dynamics studies. **A threshold value of 15cms of H2O for P_o and 8 mmHg/ml/min for R_{out} above which the patient would need shunt surgery, is suggested.**

In 7 patients the altered dynamics were present 7-10 days earlier than the clinical indication for shunting. Hence **dynamics studies would help to choose patients earlier than clinical indications**, thus can avoid further neurological deficits.

The value of dynamics studies in prediction of shunt responsiveness and its reflection on outcome was found to be to be statistically significant.

POST TRAUMATIC HYDROCEPHALUS

Post traumatic hydrocephalus has been recognised since Dandy's report in 1914. The incidence ranges from 0.7 – 86%. The differences in clinical and radiological criteria have contributed to the variations in reported incidences. Gudeman et al.[32](1981) have introduced CT criteria for diagnosing post traumatic hydrocephalus. But the accuracy of CT in differentiating post traumatic atrophic ventriculomegaly from hydrocephalus is uncertain as coated by Van Dongen et al[33](1980), Marmarou et al[27] (1996). Marmarou et al have suggested classification of these patients based on CSF dynamics studies and management accordingly.

In our study, the 8 patients in post traumatic group were classified as high pressure hydrocephalus, normal pressure hydrocephalus or atrophic ventriculomegaly according to dynamics studies, based on suggestions of Marmarou et al.[27] and managed accordingly. **62.5% of patients improved with the above management based on dynamics studies** (5 out of 8).

The head injury preceeding the hydrocephalus was subarachnoid haemorrhage and decompressive craniectomy for sub dural haematoma in the hydrocephalic group and diffuse cerebral edema and contusion brain in atrophic group. This is similar to the etiology reported by Marmarou et al in which they have found SAH in 66.7% patients with post traumatic hydrocephalus.

CONCLUSION

A Study of 50 patients with communicating hydrocephalus lead to the following conclusions:

- The improvised bolus lumbar injection method (the MIN method) used in our study has the advantage of being simple, less time consuming, requiring no sophisticated equipments, can be performed even in small hospitals, with fairly reliable and reproducible results.

- In post meningitic hydrocephalus a threshold value of P_o of 15cms of H2O and R_{out} of 8mmHg, above which patient would need shunt surgery is suggested. However a study with larger number of patients would be required for confirming this.

- In Normal Pressure Hydrocephalus the R_{out} threshold of 18mmHg for predicting shunt responsiveness and in post traumatic hydrocephalus the threshold for elevated P_o – 15 mmHg and for elevated R_{out} -6 mmHg already proposed are confirmed in our study.

- Study of CSF dynamics is a valuable tool in communicating hydrocephalus for confirmation of diagnosis, predicting shunt responsiveness, early selection of cases for shunting than clinical indications, and identifying patients who are hydrocephalus mimics like atrophic ventriculomegaly and avoid unnecessary shunt surgery.

APPENDIX - 5
POST TRAUMATIC HYDROCEPHALUS

S. No	Age	Sex	Clinical Features	Old Trauma	period from Trauma	Bifrontal Index	PVL	Ccrtical cist	Po cms of H2O	PVI	Rout mmHg	Classification	Mx	Clinical Status	Outcome GOS
1	20	M	head ache, memory, behavior disturbances	diffuse SAH	3months	0.35	no	seen	24	22.4	24.4	HPH	shunt	improved	5
2	60	M	altered sensorium, residual R hemiparesis with aphasia	L Frontal ICH with IVH	8 months	0.36	no	open	14	42	2.28	atrophy	cons	improved	4
3	55	M	E4V4 M5, Dysphasic	Multiple contusion brain	1month	0.38	no	open	14	26.8	5.55	atrophy	cons	static	3
4	45	M	E4VTM4, R Hemiparesis, h/o seizures	diffuse cerebral edema	2 months	0.35	no	open	11	33.6	2.23	atrophy	cons	static	2
5	34	M	memory disturbance, GCS 15	SAH, Contusions	2 months	0.4	no	seen	11	20.1	27.8	NPH	shunt	improved	5
6	70	F	E3V4M6, disoriented	multiple contusion brain, diffuse cerebral edema	15days	0.41	present	open	11	35.7	5.7	atrophy	cons	improved	5
7	30	M	head ache, residual L hemiparesis and dysphasia	R FTP SDH, decompressive craniotomy done	7 months	0.44	no	effaced	39	23.6	15.9	HPH	Shunt	improved	4
8	47	M	E4V1M3, seizures	multiple contusion brain, diffuse cerebral edema	6months	0.35	no	open	11	34.2	5.57	atrophy	cons	Expired	1

APPENDIX 6

IDIOPATHIC NPH - DIAGNOSTIC GUIDELINES:
Classification: Probable, possible, and unlikely categories
Probable INPH
The diagnosis of Probable INPH is based on clinical history, brain imaging, physical findings, and physiological criteria.
I. History
a. Insidious onset (versus acute)
b. Origin after age 40 yr
c. A minimum duration of at least 3 to 6 mo
d. No known causes of secondary hydrocephalus
e. Progression over time
f. No other neurological, psychiatric, or general medical conditions that are sufficient to explain the presenting symptoms
II. Brain imaging
A brain imaging study (CT or MRI) performed after onset of symptoms must show evidence of
a. Ventricular enlargement not entirely attributable to cerebral atrophy or congenital enlargement (Evan's index _0.3 or comparable measure)
b. No macroscopic obstruction to CSF flow
c. At least one of the following supportive features
1. Enlargement of the temporal horns of the lateral ventricles not entirely attributable to hippocampus atrophy
2. Callosal angle of 40 degrees or more
3. periventricular signal changes not attributable to microvascular ischemic changes or demyelination
4. An aqueductal or fourth ventricular flow void on MRI
Other brain imaging findings may be supportive of an INPH diagnosis but are not required for a Probable designation
1. A brain imaging study performed before onset of symptoms showing smaller ventricular size or without evidence of hydrocephalus
2. Radionuclide cisternogram showing delayed clearance of radiotracer over the cerebral convexities after 48–72 h
3. Cine MRI study or other technique showing increased ventricular flow rate
4. A SPECT-acetazolamide challenge showing decreased periventricular perfusion that is not altered by acetazolamide
III. Clinical
Gait/balance disturbance plus at least one other area of impairment in cognition, urinary symptoms, or both.
With respect to gait/balance,
at least two of the following should be present and not be entirely attributable to other conditions
a. Decreased step height
b. Decreased step length
c. Decreased cadence (speed of walking)
d. Increased trunk sway during walking
e. Widened standing base
f. Toes turned outward on walking

f. Retropulsion (spontaneous or provoked)
g. *En bloc* turning (turning requiring three or more steps for 180 degrees)
h. Impaired walking balance, as evidenced by two or more corrections out of eight steps on tandem gait testing

Cognition,

there must be documented impairment by screening instrument (such as the Monumental State examination),
or evidence of at least two of the following on examination that is not fully attributable to other conditions
a. Psychomotor slowing (increased response latency)
b. Decreased fine motor speed
c. Decreased fine motor accuracy
d. Difficulty dividing or maintaining attention
e. Impaired recall, especially for recent events
f. Executive dysfunction,
g. Behavioral or personality changes

Urinary Symptoms:

One of the followingt
a. Episodic or persistent urinary incontinence not attributable to primary urological disorders
b.Urinary and fecal incontinence

Or any two of the following
a. Urinary urgency b. Urinary frequency c. Nocturia
IV. Physiological
CSF opening pressure in the range of 5–18 mm Hg (or 70–245 mm H2O) as determined by a lumbar puncture or a comparable procedure. Appropriately measured pressures that are significantly higher or lower than this range are not consistent with a probable NPH diagnosis.

Possible INPH

A diagnosis of Possible INPH is based on historical, brain imaging, and clinical and physiological criteria
I. History
a. Have a subacute or indeterminate mode of onset
b. Begin at any age after childhood
c. May have less than 3 mo or indeterminate duration
d. May follow events such as mild head trauma, remote history of intracerebral hemorrhage, or childhood and adolescent meningitis or other conditions that in the judgment of the clinician are not likely to be causally related
e. Coexist with other neurological, psychiatric, or general medical disorders but in the judgment of the clinician not be entirely attributable to these conditions
f. Be nonprogressive or not clearly progressive
II. Brain imaging Ventricular enlargement consistent with hydrocephalus but associated with any of the following
a. Evidence of cerebral atrophy of sufficient severity to potentially explain ventricular size
b. Structural lesions that may influence ventricular size
III. Clinical

Symptoms of either
a. Incontinence and/or cognitive impairment in the absence of an observable gait or balance disturbance
b. Gait disturbance or dementia alone
IV. Physiological
Opening pressure measurement not available or pressure outside the range required for probable INPH

Unlikely INPH

1. No evidence of ventriculomegaly
2. Signs of increased intracranial pressure such as papilledema
3. No component of the clinical triad of INPH is present
4. Symptoms explained by other causes (e.g., spinal stenosis)

APPENDIX 7:

OUTCOME SCALES

Black Scale for assessment of shunt outcome:

- ***Excellent* :** Resumed pre-illness activity without deficit
- ***Good*** : Resumed pre-illness activity with deficit, improved in two or more categories
- ***Fair*** : Improved but did not return to previous work, Improved in one category,
- ***Transient*:** Temporary major improvement
- ***Poor :*** No change or worsening
- ***Dead :*** Died within 6 wk of surgery or as a result of surgery

Glasgow Outcome Scale

1 – dead

2 – persistent vegetative state

3 – severe disability – conscious but dependent

4 – moderate disability – independent but disabled

5 – good recovery – mild residual effects

APPENDIX 1:

CSF Dynamics in communicating hydrocephalus

Name of the patient:

Age/sex:

Ward, Unit:

Address: Ph No. :

DOA: DO Dynamics study:

DOD: DO Surgery :

DIAGNOSIS:

History:

Clinical findings

Investigations

Radiology

Bifrontal Index

Csf studies

P_o: P_p: P_t: t: rV:

PVI:

R_{out}:

Biochem: Cytology: Culture:

Diagnosis with sub classification (if any):

Management:

Reason for choosing the above management:

Status at discharge:

Follow up:

Outcome:

Remarks:

APPENDIX 2:

PATIENT CONSENT FORM

STUDY TITLE:

Study centre : Department of Neurosurgery, MMC, Chennai – 600003.

Patient's name :

Patient's age :

Identification No:

Patient may check [ ] these boxes

I confirm that I have understood the purpose of this study. I had the opportunity to ask the questions and all my questions and doubts were answered to the best of my satisfaction. [ ]

I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without my legal right being affected. [ ]

I understand that the ethical committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to

this access, however, I understand that my identity would not be revealed in any information released to the third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. [ ]

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully cooperate with the study team, and to immediately inform the study staff if I suffer from any deterioration in my health or my well being or any expected or unusual symptoms.

I hereby give consent to participate in this study.

Signature/ Thumb impression of the patient:

Place:

Patient’s name and address:

Signature of the investigator:

Name of the investigator:

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